

Conclusions: Small regional abnormalities of myocardial glucose metabolism and sympathetic innervation are common in patients with idiopathic VT and are not related to perfusion defects. These structural abnormalities may represent early manifestations of a cardiomyopathy and be pathophysiologically related to the VT.

8:45

834-2 Outcome of Patient With Life Threatening Ventricular Arrhythmias

M. Kothari, B. Bagheri, S. Hassen, A. Verma, M. Cordero, W. Ahmar, F.E. Marchlinski, C. Gottlieb, J. Heo, A.F. Iskandrian. *Allegheny University of the Health Sciences, Philadelphia, PA, USA*

Background: Most patients (pts) with life-threatening ventricular arrhythmias (VA) have LV dysfunction. This study examined the outcome of 201 pts with VA who underwent electrophysiologic testing and stress SPECT perfusion imaging.

Methods: Most pts were treated with implantable cardioverters-defibrillators (\pm antiarrhythmics) and the remaining with antiarrhythmics alone. The SPECT images were quantitatively scored based on the presence and extent of perfusion abnormality. Predictors of outcome were examined using Cox survival analysis of clinical data, ejection fraction and SPECT images.

Results: Of 159 pts (M/F 159/42, 65 \pm 10 yrs), 51 died (17%/yr) during a mean follow-up of 3 yrs. Independent predictors of survival were age, beta blocker therapy, ejection fraction and SPECT score. The presence of ischemia was not a predictor (survival 74% with ischemia and 75% without, P NS). Multivariate Cox survival analysis identified the SPECT score as the only independent marker of survival, the pts were divided into high (n=105) and low (n=96) risk groups based on SPECT score. The 3 year survival was 64% in the high risk and 86% in the low risk groups, (relative risk = 2.4, 95% CI = 1.4-4.2, P:0.0001). The SPECT score was equally predictive of survival in men and women.

Conclusion: The perfusion pattern by SPECT imaging is an important predictor of outcome in pts with VA. Those with high risk SPECT score have 2.4 fold higher death rate than pts with low SPECT score despite ICD therapy.

9:00

834-3 Fragmentation of Signals Within the QRS-Complex Detected by Magnetocardiography

R. Agrawal, P. Godde, M. Oeff, K. Czerski, H.-P. Müller, P. Endt, L. Trahms, H.-P. Schultheiss. *Dept. of Cardiology, Klinikum Benjamin Franklin, Freie Universität, Berlin, Germany; 'Physikalisch Technische Bundesanstalt Berlin, Germany*

Background: Non-invasive identification of patients with coronary heart disease (CHD) who are at high risk for ventricular tachyarrhythmias remains an unsolved problem. We hypothesize a direct pathophysiological correlation between increased intra-QRS fragmentation and the arrhythmogenic substrate and thus the risk of sudden death.

Methods: Magnetocardiography (MCG) was applied as a diagnostic tool for 41 patients (36 males) with invasively documented CHD and inducible ventricular tachycardia/ventricular fibrillation (VT/VF) during electrophysiological investigation, 41 patients (31 males) after myocardial infarction without history of VT/VF and 23 healthy subjects (16 males). A 49-channel gradiometer of the 1st order in a shielded hospital environment was used. Signals were bandpass filtered between 37 and 90 Hz. Algorithm: A fragmentation index (FI) was defined as a product of the number of extremes (E) and the sum of the amplitudes of pathlength (PL) between extremes ($FI = nE \cdot \Sigma PL$).

Results:

	CHD with VT/VF	Post MI	Control Group
n	41	41	23
Age	61.2 \pm 10.5	61.5 \pm 8.8	41.7 \pm 13.7
EF	47.0 \pm 17.7	56.8 \pm 13.0	-
FI	73.0 \pm 37.9	48.7 \pm 14.0	42.4 \pm 7.4

Conclusion: In the MCG, patients with VT/VF and CHD show a significantly increased fragmentation within the entire QRS-complex ($P < 0.05$). A high FI seemed to be a specific sign for an arrhythmogenic substrate.

9:15

834-4 Demonstration of Intraventricular Conduction Block in Patients Late After Myocardial Infarction With Ventricular Tachycardia Using Three-dimensional Electroanatomic Mapping

D. Pfoiffer, P. Kluge, T. Walter, A. Neugebauer. *Department of Cardiology, University of Leipzig, Germany*

Decremental conduction is known to be a prerequisite for reentrant arrhythmias.

The aim of the study was to demonstrate localization and rate-dependency of abnormalities in conduction in the left ventricle in patients (pts) late after myocardial infarction (MI) and ventricular tachycardia (VT) using the CARTO[®] system.

Patients and Methods: Left ventricular mapping during right ventricular pacing with cycle lengths of 600, 500 and 400 ms has been performed in 6 patients (6 male, age 64 \pm 4 y) after MI using the CARTO[®] system. No patient had rate-dependent bundle branch block. Identical mapping points in the left ventricle were collected after 30 s of pacing. Points were eliminated if they could not be accepted in any of the three maps per patient.

Results: There were 18 maps with 13-33 points/map. The area of MI could be detected by identical conduction delay and block independent of pacing rate in all three maps per patient. Rate-dependent development of conduction delay in septal and anterior area was seen in the 400 ms maps in comparison to 600 and 500 ms maps. These areas with decremental conduction were found as in perinfarction localization (n = 2) as in the septal area (n = 5). More than one area of rate-dependent delay and block was seen. The decrement ranged between 45 and 65 ms.

Conclusions: The CARTO[®] system can be used for demonstration of rate-dependent conduction delay and block in patients late after MI. Rate-independent block represents the infarcted area. Rate-dependent block is localized in septal and perinfarction areas as a prerequisite for reentrant VT.

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834-5 Structural Abnormalities Defined by Three-Dimensional Echocardiography in Patients With Right Ventricular Tachycardia

R.T. Hahn, S.M. Markowitz, A. Ilterci, K.M. Stein, R.B. Devereux, B.B. Lerman. *New York Hospital-Cornell Medical Center, New York, NY, USA*

Background: In patients with right ventricular tachycardia, conventional 2-dimensional transthoracic echocardiography has limited sensitivity for detecting subtle abnormalities of the right ventricle (RV). We performed 3-dimensional (3-D) echocardiograms to determine the utility of this technique in characterizing RV abnormalities in this group of patients.

Methods and Results: Six patients with either arrhythmogenic RV dysplasia (ARVD) or idiopathic VT arising RV outflow tract (RVOT) had 3-D echos constructed using a TomTec Echo-Scan. In 4 patients, the diagnosis of ARVD was based on invasive electrophysiologic studies, myocardial biopsy, surface ECG and/or magnetic resonance imaging (MRI). Two patients with idiopathic VT demonstrated inducible ventricular tachycardia (VT) with a left bundle branch block configuration that was sensitive to adenosine. 3-D echo demonstrated significant structural and functional RV abnormalities in all patients with ARVD. These findings included marked global or regional dilatation of the RV in 3 patients, focal hypokinesis or dyskinesis of the inferior, anterior or apical RV walls in 4, and focal thinning of the apex in 1 patient. In patients with idiopathic VT, one showed thinning and akinesis of the inferior wall of the RV, and the other was normal. MRI findings were confirmatory in 4/5 patients.

Conclusions: In patients with ARVD, 3-D echo identified regional abnormalities in all patients. In addition, some patients with idiopathic VT may demonstrate similar structural abnormalities.

9:45

834-6 Feasibility of Virtual Anatomy-based Three-dimensional Mapping in a Canine Model

D.L. Packer, S. Aharon, S.B. Johnson, J.J. Camp, R.A. Robb. *Mayo Foundation, Rochester, Minnesota, USA*

The ablative approach to most cardiac arrhythmias is dependent on an understanding of both underlying anatomy and characteristic local tissue activation in critical myocardial regions. Defining this target anatomy with fluoroscopy is difficult, and current three-dimensional mapping utilities create only approximations of underlying anatomy or are limited in their capability of defining spatial boundaries. To test the feasibility of creating a four-dimensional mapping capability based on actual underlying cardiac anatomy, 3 dogs underwent activation mapping and complete cardiac imaging. Three-dimensional anatomy was established either by one CT scanning with imaging at 1-3 mm intervals or with 360°, stepped, 7.5 MHz phased-array, long axis imaging of the intact canine heart. Custom software was designed to produce anatomically accurate three-dimensional, geometric computer images of epicardial and endocardial targets. Cardiac electrical activation as a fourth dimension, acquired using a 56 channel epicardial mapping sock and simultaneous 25-32 channel intracardiac baskets, was superimposed on the three-dimensional surface models. Electrogram correlation was established through imaged electrode/waveform matching. Maps generated off-line clearly demonstrated anatomically accurate electrical activation that could be manipulated in a virtual reality environment. Although preliminary, these data demonstrate the feasibility of creating four-dimensional activation

maps based on actual underlying anatomy. Additionally, this approach aided substantially in conceptualizing target atrial structures. With additional development, an on-line tool for use in guiding ablation of cardiac tissue is now plausible.

835 Hypertrophic Cardiomyopathy: Clinical Course, Ablation, and Pacing

Tuesday, March 31, 1998, 8:30 a.m. - 10:00 a.m.
Georgia World Congress Center, Room 367W

835-1 Hypertrophic Cardiomyopathy in the "real World": Prevalence, Demographics and Clinical Expression in an Unselected and Unreferred Hospital-based Patient Population

B.J. Maron, L.C. Poline, S.A. Casey. *Minneapolis Heart Institute Foundation, Minneapolis, MN, USA*

Background: Hypertrophic cardiomyopathy (HCM) is the most common cause of sudden death in the young, including competitive athletes. Establishing the prevalence and clinical profiles of HCM in various populations is crucial to achieving a true perception of its clinical impact. Prevalence of HCM in a general (non-hospital-based) population has been previously reported as 0.17%.

Methods: In this study, we assessed occurrence and clinical features of HCM in an unselected pt cohort not subject to tertiary center referral bias.

Results: Between 1980 and 1995, 131,545 cardiac pts were evaluated; 221 (0.17%; 1,711,000) proved to have HCM. Age at diagnosis was 4-87 years (mean 50 ± 18); 121 (55%) were male. Left ventricular (LV) wall thickness was 15-38 mm (mean 22 ± 5). Hypertrophy was most commonly diffuse, in 2 or 3 LV segments (135 pts; 61%), but was localized to only 1 segment in 86 pts (39%). Outflow obstruction (gradient >30 mm Hg) was present in 51 pts (23%). Most pts (129 or 58%) had no or only mild symptoms; 92 (42%) had moderate-severe symptoms.

Conclusions: In an unselected hospital-based pt population evaluated for heart disease, the prevalence of HCM was 1:500 (0.17%) identical to its occurrence in the general population. In a "real-world" clinical setting, most HCM pts have little or no symptoms, unlike tertiary centers in which most pts are severely symptomatic.

8:30

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835-2 Long-term Outcome of Hypertrophic Cardiomyopathy With Mid-Ventricular Obstruction

A. Woo, E.D. Wigle, R.C. Rakowski, S.C. Siu, H. Rakowski. *The Toronto Hospital, Toronto, Canada*

Background: Hypertrophic cardiomyopathy (HCM) with midventricular form of HCM whose long-term outcomes are unknown.

Methods: Forty-one pts with HCM and MVO underwent serial clinical and echo-Doppler assessments. There were 19 men (44%). Mean age was 37.3 years (range, 11-67 years). The mean follow-up was 8 years (range, 1-27 years).

Results: Presenting features were dyspnea (83%), angina (70%), syncope (18%), and a long diastolic murmur (51%). The latter was associated with a diastolic mid-cavity gradient. A family history of HCM was present in 44% with non-MVO forms of HCM predominating. The mean MVO gradient was 50 (range, 25-112) mmHg. Associated LV outflow tract obstruction (LVOTO) was present in 15 pts (37%) with a mean gradient of 59 mmHg.

Follow-up: Atrial fibrillation was uncommon (12%) with no thromboembolic events. Sustained ventricular tachycardia (VT) occurred in 2 patients (5%) and only in association with an apical infarction. Dual chamber (DDD) pacing for MVO resulted in mean gradient reduction of 52 to 32 mmHg in 3/4 pts (75%). Surgical myectomy decreased mean MVO gradient (40 to 10 mmHg) in 6/9 pts but increased mean MVO gradient (29 to 56 mmHg) in 3 pts when LVOTO alone was relieved. There were 3 cardiac deaths, 1 following myectomy and 2 related to apical infarction and VT.

Conclusions: HCM with MVO has a relatively benign prognosis in the absence of apical infarction. Treatment with DDD pacing or myectomy is only of variable benefit.

835-3 Acute Results of Catheter Treatment in Hypertrophic Obstructive Cardiomyopathy

H. Seggewiss, L. Faber, U. Gleichmann, D. Fassbender, S. Strick. *Dept. of Cardiology, Heart Center NRW, Ruhr-University Bochum, Bad Oeynhausen, Germany*

Background: Percutaneous transluminal septal myocardial ablation (PTMSA) seems to be a promising treatment option in symptomatic pts. with HOCM. We report on the acute results in 83 pts.

Methods: 83 symptomatic pts. (41 men; age 54.2 ± 14.4 years; 5 pts. with prior myectomy; 6 pts. with DDD-pacer; 32 pts. with prior syncope; NYHA class 2.7 ± 0.6) were treated. LVOT gradients (LVOTG) were simultaneously measured at rest and post extrasystole (ES). 1.1 ± 0.4 (1-3) septal branches were occluded by injection of 3.5 ± 1.6 ml absolute alcohol during balloon dilatation of the proximal part of the septal branch with an over the wire balloon catheter.

Results: LVOTG reduction was achieved in 77 (94%) pts.: complete in 21 (25%), >50% in 49 (58%), and 20-49% in 8 (10%) pts. LVOTG were reduced from 74.4 ± 35.1 to 17.3 ± 20.0 mmHg at rest (p < 0.00001) and from 147.3 ± 42.3 to 62.3 ± 45.5 mmHg post ES (p < 0.00001). Pts. with echo guided identification of the target septal branch (n = 53) had a higher LVOTG reduction (79 ± 24% vs. 64 ± 38%; p = 0.04). 54 (65%) pts. developed trifascicular block, requiring temporary (n = 43) or permanent (n = 11; 13%) pacemaker. 43 (52%) pts. had new -predominantly right - bundle branch blocks. Maximal CK rise was 667 ± 331 U/L. 2 (2.4%) pts. died from ventricular fibrillation (day 9) or pulmonary embolism (day 2).

Conclusion: PTMSA of HOCM is a promising treatment options in symptomatic pts. Careful monitoring during hospital stay is necessary because of the potential risks of the therapeutic infarction. Long-term follow-up and comparison with conventional treatment is necessary in order to estimate its definitive therapeutic significance.

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835-4 Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy: Clinical and Non-Invasive Follow-up Results

H. Seggewiss, L. Faber, U. Gleichmann, S. Strick, P. Ziemssen. *Dept. of Cardiology, Heart Center NRW, Ruhr-University Bochum, Bad Oeynhausen, Germany*

Background: Percutaneous transluminal septal myocardial ablation (PTMSA) by alcohol induced septal branch occlusion in pts. with HOCM results in acute reduction of left ventricular outflow tract gradients (LVOTG) in 90% of the pts. Mid- and long-term follow-up results are unknown.

Methods: 49/53 symptomatic pts. (26 men; age 51.3 ± 15.0 years; 3 pts. with prior myectomy, 21 pts. with prior syncope, NYHA class 2.7 ± 0.6) showed significant reduction of LVOTG after PTMSA. Mean LVOTG were reduced from 58.9 ± 43.3 to 28.5 ± 26.6 mmHg at rest and from 142.7 ± 53.9 to 70.7 ± 53.3 mmHg at stress (each p < 0.00001). 7 (13%) pts. required DDD-pacemaker (PM) due to permanent trifascicular block. We report on 3 months follow-up in 53 pts. and 1 year follow-up in 12 pts. after PTMSA.

Results: No cardiac complications were observed during follow-up. After 3 months 49 (92.5%) pts. showed clinical improvement (NYHA 1.2 ± 1.0; p < 0.00001) with an increase of workload from 90.7 ± 62.0 to 114.0 ± 41.4 Watts (p < 0.01) - the results were comparable in pts. with and without DDD-PM. 4 pts. underwent successful re-PTMSA. 29 (55%) pts. showed further LVOTG reduction. Compared to acute results mean LVOTG decreased to 19.2 ± 18.2 mmHg at rest (p < 0.001) and to 58.7 ± 47.6 mmHg at stress (p = 0.03). Furthermore, we observed significant reduction of septal (21.5 ± 3.7 to 17.7 ± 3.4 mm; p < 0.0001) and left posterior wall thickness (14.2 ± 2.5 to 13.2 ± 2.1 mm; p < 0.001) as well as decrease of pulmonary artery pressure at max. workload (43.4 ± 11.9 vs. 39.9 ± 10.1 mmHg; p = 0.04). After 1 year 5/12 (42%) pts. showed further LVOTG reduction and ongoing symptomatic improvement (NYHA 1.2 ± 1.2).

Conclusion: After PTMSA pts. showed ongoing symptomatic improvement. Remodeling after circumscribed septal infarction results in further LVOTG reduction in >50% of the pts.

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835-5 Acute and Long-term Results After Transcatheter Ablation of Septum Hypertrophy in Hypertrophic Cardiomyopathy

F.H. Gietzen, H.J. Kuhn, C.J. Leuner, J. Hegselmann, C. Strunk-Mueller. *Dept. of Cardiology, Bielefeld Hospital, Bielefeld, Germany*

We report on 67 transcatheter ablations of septum hypertrophy (TASH) in